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EDITORIAL

In this issue of the Journal will be found abstracts of a few articles of interest to students, recently published by members of the staff of the Faculty of Medicine. It is hoped that the publication of abstracts will become an established feature of the Journal. The reasons for inaugurating this feature are at least two in number. The first and most important reason is the hope that these abstracts may lead the students to a better knowledge of the type of information which is available in the current periodicals. If every student realized how much of his studying can be done from journals there would be far less dependence upon notes and text-books. We have no quarrel with the use of notes and text-books but, since the student will have to begin keeping up-to-date as soon as he graduates, we feel that he should early become acquainted with the journals which will form one of his chief connections with medical progress after graduation.

The second, and scarcely less important reason for publishing abstracts, is to impress upon the students the great amount of clinical and laboratory re-

search that is being done by the members of our own staff, and the valuable contributions to knowledge which they are making. We feel that this knowledge will not only increase our pride in the Faculty but will also make more clear the changing nature of much of the material that we are taught. Every student, in considering any medical subject, should be interested primarily in the views of his own teachers upon that subject. Very often students are not aware of excellent papers written by members of our own staff. These abstracts are only a part of a scheme to remedy this defect. Another part of the scheme is the posting, in the Medical Reading Room, of a list of staff articles appearing in the periodicals received here. If the students show sufficient interest in this scheme it is hoped that a reprint library and possibly also a card-index of staff publications will be added to the library.

While discussing the use of periodicals added to the library it might be well to point out the many libraries which are available for student use. The Medical Reading Room is, of course, familiar to all. In it are most of the commoner medical and scientific journals. There are, however, several less well known libraries which are open for the student use. Among these may be mentioned the library of the Connaught Laboratories in the School of Hygiene Building, the library of the Department of Physiology in the Medical Building, the Anatomy library in the Anatomy Building and the library of Pathology and Bacteriology in the Banting Institute. Each one of these contains many special periodicals and some books not to be found in the Medical Reading Room. The excellent library of the Academy of Medicine at 13 Queens Park is also available for use by senior students upon presentation of a letter of introduction from a member of the Academy.

It will be noticed that the majority of the abstracts appearing in this issue are from papers published in the Canadian Medical Association Journal. Although many students already subscribe to that excellent Journal there are still some who do not realize its value and it is for those students that this group of abstracts is published. Every medical student should subscribe to, and read, the Canadian Medical Association Journal both as an undergraduate and throughout his professional career.

Another way in which the student can keep abreast of scientific progress is by atending the many scientific meetings which are held in the University and at the various hospitals. There are several scientific societies which welcome students at their meetings; among these may be mentioned the Toronto Physiological Society, the Toronto Biochemical Society, and the Academy of Medicine. Students are also permitted to attend clinical conferences in some of the hospitals. Meetings of this kind are often very stimulating and every student should attempt to attend those in which he is particularly interested.

It was a great pleasure to see the name of J. D. Galbraith in the Honour List in the current issue of the Diplomate. Dr. Galbraith, who graduated from Toronto in 1935 obtained an average mark of 90.50 per cent. at the September examinations for Part II of the National Board Examinations in the United States. It is reassuring to see that our graduates can more than hold their own in competition with the best trained men from other colleges. We extend the congratulations of the undergraduates to Dr. Galbraith.

THE DIAGNOSIS OF BONE TUMOURS

By R. I. HARRIS, M.C., M.B., F.R.C.S. (C.).
Department of Surgery.

Though bone tumours are comparatively rare they are of such serious significance as to demand the closest scrutiny. Our only hope of cure lies in early recognition and radical excision. Hence diagnosis is of the utmost importance.

Classification of Bone Tumours.

It is well to bear in mind the various types of bone tumours which may be encountered. These can be classified into two great groups, benign and malignant.

Benign.

Giant-celled tumour.
Osteochondroma.
Enchondroma.
Bone Cyst.

Malignant.

Osteogenic sarcoma.
Ewing's tumour.
Periosteal fibrosarcoma.
Multiple myeloma.
Metastatic tumours.

In addition we must bear in mind certain lesions which may resemble bone tumours and which may cause difficulties in diagnosis.

Lesions Which Sometimes Resemble Bone Tumours.

Fibrocystic disease.
Myositis ossificans.
Osteomyelitis.
Certain forms of tuberculosis.
Syphilis of bone.
Paget's disease of bone.

Four Important Elements in Diagnosis.

In attempting to reach a diagnosis in any case in which bone tumour is suspected much information can be obtained by consideration of four important points which we may put down here in the form of questions.

1. Is the lesion single or multiple?

The malignant tumours with the exception of multiple myeloma are all single in their origin. Metastases occur most frequently in the lungs, rarely in other bones. The benign tumours may exist as single lesions but very frequently are multiple.

2. How old is the patient?

These is a very marked tendency for bone tumours to arise at definite age periods. While this rule is not infallible it is often of great help in the diagnosis.

3. Where in the skeleton is the tumour located?

Bone tumours tend to occur at specific sites in the skeleton. Not only do certain tumours have a predilection for certain bones but often for a specific site in the bone. Thus giant-celled tumours occur only in the epiphysis (usually of lower end of radius, lower end of femur or upper end of tibia). Osteogenic sarcoma occurs in the ends of the diaphysis and does not involve the epiphysis.

4. What is the X-ray appearance?

This is the most important single aid to diagnosis short of biopsy. The X-ray appearance of most bone tumours is distinctive.

Bearing these four points in mind we may summarize briefly the characteristics of the more important lesions.

Osteogenic Sarcoma.

1. Commences as a single lesion and metastasizes to lungs commonly, rarely to other bones.

2. *Age Incidence.*—With one important exception it is a disease of adoles-

cence and young adult life. The exception is the type of osteogenic sarcoma which is implanted upon Paget's disease of bone. These occur in the fifth, sixth and seventh decades of life.

3. *Location in Skeleton.*—The common sites are the ends of long bones, but as a rarity any bone may be involved. The long bones chiefly involved are lower end of femur, upper end of tibia, and upper end of humerus. In a long bone the lesion first appears in the end of the diaphysis. Only a short extent of the shaft is involved. The epiphysis is not invaded.

4. *X-ray Appearance.*—Though this varies within wide limits it usually is distinctive. Two contrasting features distinguish the X-ray picture of osteogenic sarcoma. The first is destruction of the bone in which the tumour has arisen, by the tumour cells. The second is the formation of new bone. This new bone is of two origins. It may be formed by the tumour cells themselves —tumour bone—or it may be formed as the result of irritation of normal bone structures by the tumour cells—reactive bone. The two elements of bone destruction and new bone formation exist in the widest variety of combinations, from the case in which bone destruction only is seen (osteolytic tytpe) to that in which the bone formation is so great as to mask the areas of bone destruction (sclerosing type).

The usual X-ray picture is that of a lesion in the end of the diaphysis of a long bone. It reaches to the epiphysis but does not invade it. The common situations are lower end of femur, upper end of tibia and upper end of humerus. An area of bone destruction of greater or less extent without clear outlines occur usually to one side of the shaft. New bone is laid down outside of the line of the former cortex. This may be deposited in the form of irregular masses but often it is laid down as spicules radiating out in straight lines at right angles to the shaft. This "sun ray" appearance is very characteristic of osteogenic sarcoma and rarely occurs in any other form of bone tumour. Its appearance therefore is important. At the periphery of the tumour the periosteum is raised and beneath it is deposited a small wedge of new bone. The extent of shaft involves is small, usually about one-fifth or one-sixth of its length.

Ewing's Tumour.

1. Commences as a single lesion. Metastasizes to lungs and also to other bones.

2. *Age Incidence.*—This is a disease of childhood and adolescence. The age incidence is somewhat younger than in osteogenic sarcoma.

3. *Location in Skeleton.*—The primary lesion involves the end of the diaphysis of long bones as does osteogenic sarcoma, but a much greater length of shaft is involved, usually one-third to one-half the length. The epiphysis is not invaded. Common sites, lower end of femur and of tibia.

4. *X-ray Appearance.*—As in osteogenic sarcoma the basic changes seen are areas of bone destruction plus areas of new bone formation. The picture, however, is entirely different from osteogenic sarcoma and usually is distinctive. First, the extent of involvement of the shaft is great—one-third to one-half the length. Though the lesion usually starts in the end of the diaphysis, it may commence in the centre of the shaft. The areas of bone destruction are small and multiple, scattered throughout the involved area. New bone formation frequently obscures the small areas of the bone destruction. Certainly the new bone formation dominates the picture. The cortex of the involved bone is expanded. New bone is laid down in layers outside of the old cortex and these layers being separated from one another by areas of less density, are distinguished from one another in the X-ray. This "layered" formation of new bone is characteristic and is referred to as "onion skin appearance."

Since Ewing's tumour occurs in children and frequently is accompanied

by fever and leukocytosis, the mistaken diagnosis of osteomyelitis often is made.

Periosteal Fibrosarcoma.

This is not a true bone tumour. It is a fibrosarcoma arising from the periosteum or from fascial planes in close association with bone. It does not form bone, hence does not cast a bony shadow in the X-ray. The bone is involved late, if at all. Its importance lies in the necessity of distinguishing it from true bone tumours.

Multiple Myeloma.

This malignant tumour is unique in being multiple in its origin. It occurs in the middle decades of life, chiefly in the ribs and spine. Usually the tumours are very numerous and small in size. Occasionally the disease may first manifest itself as a single tumour and this, if it occurs in an unusual situation, such as the shaft of the femur, may give difficulty in diagnosis. The X-ray appearance is that of small punched-out areas of bone destruction.

Giant-Celled Tumour.

This is the most important of the benign tumours.

1. Commences as a single lesion.
2. *Age Incidence*—Occurs from young adult life to middle age.
3. *Location in Skeleton.*—The usual site is in the epiphysis of a long bone or rather in that part of a long bone which formerly was an epiphysis since by the time these tumours appear, the epiphyseal lines are fused. Common sites are lower end of femur and upper end of tibia.
4. *X-ray Appearance.*—This is distinctive. The tumour destroys bone as it grows but it has the singular property of expanding the bone. In the X-ray we see a large area of bone destruction with clearly defined scalloped margins demarcating it from normal bone. Usually the whole epiphysis is involved. The original cortex is expanded sometimes to twice its normal width. It extends down to, but does not invade, articular cartilage. The scalloped margins indicate that the tumour is lobulated and this is further evidenced by the appearance of bony trabeculae which run across the wall of the tumour and separate adjacent lobulations. At a late stage the cortex may be performed and the tumour invade adjacent soft tissue.

The Value of Biopsy.

Though analysis of the facts concerning any bone tumour carried out in the manner outlined will usually permit one to make an accurate diagnosis, there still occur many cases in which diagnosis is difficult. This is especially apt to be the case if the tumour occurs in an uncommon situation or if the X-ray appearance simulates that of some other form of bone tumour. Many such cases occur. For this reason actual histological examination of the tumour tissue is often the only means of making a diagnosis. Also it is by all odds the most accurate. With all the aids to clinical diagnosis backed by mature experience, the percentage of error in diagnosis is sufficiently high to make biopsy important. At the Dunlap Institute it is the custom to perform a biopsy upon every case of accessible bone tumour as a final step in the diagnosis. One should realize, of course, that even pathologists have difficulties and a certain degree of error is possible even where the tissue is available for examination under the microscope. Nevertheless biopsy is at present our most accurate means of diagnosis and corrects an error in diagnosis with sufficient frequency as to justify its use in every case of bone tumour.

DUODENAL SPASM

E. E. CLEAVER, B.A., M.B., F.R.C.P (C.)

Under the captain of Duodenal Spasm are placed first, those cases which present the symptoms of duodenal ulcer, but with negative X-ray findings, and secondly, those cases which present the duodenal picture, with X-ray findings of reflex spasm of the duodenal cap.

In the first group the symptoms complained of are definitely duodenal in type and are generally indistinguishable from the symptoms of a frank ulcer. The patient complains of epigastric pain of a burning, gnawing or hunger type, coming on from one to four hours after meals, relieved by the taking of food or an alkali. These symptoms come on, in attacks that may persist for from one to six weeks, and then there is complete absence of symptoms for weeks, months, and sometimes years. Nervous symptoms such as headache, dizziness and fatigue, palpitation and dyspnoea are sometimes present. A large percentage of this group is emotionally unstable. Apprehension, anxiety, anger, worry and fear may initiate an attack. A certain number of these cases may have symptoms only for a few days, and it is unusual to have pain during the night.

The cases of reflex duodenal spasm present the same picture as the first group, but in addition a history is obtained which suggests a diseased appendix, gall bladder, inflammation of the colon, etc. It is possible that diseases in the chest, genito-urinary, and other systems may be responsible for reflex duodenal symptoms. To cite a typical case: A young man of 30 years of age complained of having attacks of pain in the spring and fall. The symptoms were strongly suggestive of duodenal ulcer—epigastric pain coming on two or three hours after meals, and having characteristic food ease. The gnawing pain did not last more than two or three days and there was then freedom from symptoms for several months. Gastric analysis showed a high acid curve. The examination of stool was negative for occult blood. X-ray examination showed a filling defect in the duodenal bulb, but no active crater was visualized. A long, tender, kinked and fixed appendix was seen. After repeated attempts a history of an acute attack of pain in the right iliac fossa, associated with vomiting was obtained. The pain lasted for only one day. An X-ray repeat after giving fairly heavy doses of atropine showed a normal duodenal cap and the same kinked appendix. Operation was advised. After opening the abdomen a pathological appendix with firm adhesions was removed. Examination of the duodenum showed no evidence of an active or healed duodenal ulcer.

An analysis of 300 cases presenting the duodenal syndrome revealed the fact about 100 cases gave a typical history, but on roentgenolical examination there was no evidence of duodenal ulcer. Of these 100 cases, 85 were placed in the group of duodenal spasm, as no intrinsic cause was found. In 15 cases an apparent cause for the reflex spasm was found in a diseased appendix, gall bladder, colon, etc. This group of 85 which is placed under the heading of duodenal spasm should be be regarded as potential ulcers. Some of these cases with typical duodenal ulcer history have been followed for years, and repeated X-ray examinations have been negative, until finally a positive X-ray finding is obtained.

In contrast, many cases have been followed for years, with frequent X-ray examinations and up to the present time no ulcer has been demonstrated.

A young man of 26 has been follow-

ed for four years. He gave a very suggestive duodenal history. Three X-ray examinations at intervals of about a year were entirely negative, but the fourth examination was positive for duodenal ulcer.

These duodenal spasm cases represent about one-third of those presenting duodenal symptoms, but with an demonstrable ulcer. This group should be placed on a duodenal regimen. Emphasis should be placed on the importance of mental and physical fatigue. These individuals should have a midday rest, and at least eight or nine hours in bed at night. Moderate exercise should be encouraged. Alcohol should be eliminated. Tobacco, if used at all, should be strictly limited. Teeth and tonsils should be carefully watched. Regularity in going to stool is essential. If constipated, Liquid Paraffin or Paraffin and Agar may be used. A bland balanced diet should be followed, with emphasis on the importance of taking a glass of milk or buttermilk, or soda biscuits, between meals and at bedtime. Raw fruits, raw vegetables, twice cooked meats, and any article of food which has a large residue should be avoided. An alkaline mixture is not necessary if the symptoms can be controlled by the diet. It is important that the patient should return at stated intervels for a review by the physician and an X-ray re-check.

Conclusion.

In conclusion, if these cases of potential ulcer, which are grouped under the heading of Duodenal Spasm, are placed on careful duodenal ulcer treatment, the majority of them will not develop a frank ulcer.

INFECTION AND TRAUMA AS CAUSES OF MENTAL DISEASES

By W. R. FEASBY, B.A., '37.

In classifying the organic lesions thought to be the cause of mental disease, it seems convenient to follow the very familiar mnemonic of the etiology of disease, viz.:

I: Congenital.
II: Infection.
III: Trauma.
IV: Neoplastic.
V: Deficiency and Metabolic Disorders.

It is the purpose of this paper to collect some of the recent opinions concerning the part played by the infection and trauma in the production of mental disease.

By mental disease we shall mean any recognized pathological deviation from a normal or standard behaviour. We shall consider as causally related only those conditions known definitely to have a direct and controlled temporal relation.

A: Infection.

Inflammation is the basic pathological effect of infection and may be considered as chronic or acute.

The effects of the chronic inflammation due to syphilis and tuberculosis are well known to most students of medicine. We need only comment, therefore, on the typical neuro-syphilitic who goes happily, often very grandiosely, to a certain, imminent death, and on the patient at the end-stage of tuberculosis, when, with degeneration of central nervous tissue, an euphoria is frequently observed.

There have been some recent notes in the literature on the disseminated or

diffuse scleroses. One hesitates before assigning a chronic inflammatory process as the fore-runner of the lesion, which is a progressive plaque-like degeneration of the central nervous system. Charcot included in his famous but infrequent triad of disease "scanning speech," the cardinal sign, which is exemplary of the first great group of mental signs arising in this condition, namely:

(i) *Speech difficulties* — scanning speech; various aphasias. These are associated with plaques in the language zone.

(ii) *Memory loss and confusion,* which are associated with lesions of the frontal lobe.

(iii) *Emotional incontinence*:— explosive laughter; crying, associated with lesions of the mid-brain.

A second group of signs of mental disease due to disseminated sclerosis would seem to be associated definitely with lesions but with super-added functional factors, e.g.:

(i) *Ideas of sin*—as compensation for persistent priapism due to cervico-dorsal plaque localization.

(ii) *Irritability, instability*—following lesions of the bladder control tracts.

(iii) *Forced gaiety, neurotic tendencies*—following muscular weakness resultant from localized motor cell plaque lesions.

Another poorly-known disease in which chronic inflammation is the pathological process, is Alzheimer's disease, which results in cerebral scarring, diffusely scattered through the brain. It is a pre-senile lesion, possibly of vascular origin. Patients develope extreme irritability, may have atypical epileptic seizures, and go slowly to an early grave. Possibly, another variation of this disease process is the chronic endarteritis followed by cerebral degeneration, witnesses so frequently in the aged, who may be described as the slobbering, blearly-eyed, emotionally incontinent, "lean and slippered pantaloon, sans teeth, sans eyes, sans everything!"

The acute infections are an infrequent but, disastrous cause of mental disease, for acute encephalitis, influenzal complications, erysipelas, and others may cause permanent destruction of personality. The association for research in nervous and mental diseases presents the following useful classification of acute reactions with demonstrable organic lesions as a basis:

(i) *Acute delerium.*

(ii) *Mental torpor*:—dullness; somnolence; stupor; comma.

(iii) *Amnesia*:—confabulatory syndrome.

(iv) *Personality destruction.*

Of the acute infections, encephalitis lethargica (or epidemic encephalitis) has received the most attention, because of the serious behaviour problems of its unfortunate victims. To see one of these children moving about restlessly, playing impish, disconcerting pranks, continuously a source of trouble in the home, makes one realize the seriousness of infection as a cause of mental disease. The common psychotic reactions observable in this disease are:

(i) *Sleep disturbance.*
(ii) *Delirium.*
(iii) *Emotional inconsistence.*
(iv) *Motor signs*:—fantastic posture.

Of these signs, only emotional incontinence remains at the end of the acute stage, but it may lead to:

(i) *Conduct disorder in children.*
(ii) *Neurasthenia or psychasthenia.*

At autopsy, the pathological findings, while not always typically related to the psychotic reactions, seem to be significant, e.g.,:

(i) *Acute inflammation* involving cortex and medulla.

(ii) in late stages—*haemorrhage* into peri-vascular spaces.

(iii) *Minimal vascular lesions.*

Whatever may be subsequently determined, it seems certain now that these behavious reactions follow directly upon the acute inflammatory process, as a reaction to the virus, and are the result of the lesions.

B: Trauma.

With the ever increasing incidence of head injuries, despite safety glass and courtesy codes, brain trauma becomes an interesting scientific and serious economic problem. Suppose, for example, that Mr. Brown runs his car into a cement pillar and receives a crushing injury to his left frontal region, resulting in a cranial fracture and laceration of the brain. He is treated by brilliant surgeons and appears to recover. But, six weeks later, he developes a mental illness, is incapacitated and seeks compensation on his insurance policy. Then, begins the long battle in the courts, which is essentially the same as that in scientific circles, viz.: Is Mr. Brown's mental disease due to the injury received in the accident or is it due to the human weakness for compensation? There are authorities to support each side of the argument and some believe that both factors are involved.

There are some rather striking case reports in the current literature which support the view that trauma to a fairly large area of cerebral tissue results in marked deterioration of personality. The American Crow-Bar case, in which loss of both frontal lobes resulted in extreme irritability and loss of ability to direct a gang of men, is well known by most medical students.

Dandy has published reports on a number of cases of similar type. The most striking is that of a man in whom both frontal lobes were removed because of tumor. This patient has been described as "an extravagant beast" devoid of human emotions, but able to carry on an animal existence.

Dandy, 1933, reports three cases of complete personality change and loss of memory were treated by removal of largely diseased right hemisphere and seemed to show complete recovery of their normal personality.

Gardner, 1933, reports three similar cases in which the one patient surviving operative removal of the right cerebral hemisphere showed recovery from the mental illness. Therefore, Gardner believes that the frontal lobe is responsible for the personality.

Zollinger reports that removal of the left frontal lobe in a right-handed woman resulted in relief of signs of emotional disturbance resultant upon destruction of tissue by tumor.

Dr. K. G. McKenzzie, speaking recently, reported these cases:

Case i.—Removal of a quarter of both frontal lobes following severe injury to a boy 19 years old resulted in temporary loss of restraint of sexual impulses and in lasting (two years) loss of ambition and inability to shoulder even light responsibilities.

Case ii.—Left frontal lobectomy in a girl aged 16 years with brain abscess has resulted in epileptic fifits due to scarring affecting the motor cortex. Now, at 21 years of age, she feels that her memory, while good, is impaired. However, there has been no loss of ambition in this case.

Case iii.—A man was hit on the back of the head by thugs six years before death. Following his injury, he showed marked loss of ability to work at his job of book-keeping and was much more irritable and difficult with people. At autopsy, the brain showed extensive scarring of the inferior surface of both frontal lobes.

Therefore, these cases seem to indicate that gross cerebral trauma or surgical results in mental disease, characterized by:

(i) *normal cognition.*

(ii) *complete loss of conation.*

(iii) *complete destruction of normal emotion,* usually with extreme irritability. However, it has been estimated recently that head injuries, brain tumors, abscesses and trauma accounts for only ½ to 1% of all fresh admissions to mental hospitals.

But, these cases do not account for all admissions to mental hospitals in which trauma may be a factor for concussion and contusion may be very important in this regard. Indeed, a summation of minor traumas may well cause

permanent damage to certain areas of cerebral cortex and medulla. The brain is a very soft organ within the hard cranium. Therefore, jarring of severe degree to the cranium results in injuries of the "contra coup" type, usually resulting in death.

But often the jarring is less severe, for example, sudden falls, striking the head on a beam, or jolts on the rugby field, and as yet we have little pathological material with which to study these cases. It is known that relatively mild head injuries to neuro-syphilitics and chronic alcoholics may cause severe mental symptoms. In addition, the belief that the milder type of injuries may result in a less marked type of mental disease with insidious onset. For example, chronic subdural haemorrhage is cited by Dr. K. G. McKenzie in this regard. Buzzard states that simple injuries may lead to anxiety neurosis when functional elements are added. Kinnear Wilson believes that the emotional disturbances that occur so frequently after brain trauma are due to paleothalmic lesions which are not necessarily localized but lead to misinterpretation of stimuli. Curran and Schilder have recently reported on paraphosias in which left subdural haemorrhage led to marked difficulty with definitions and reversion to native tongue. They have shown the relation of these speech difficulties to certain of the speech abnormalities seen in schizophrenia. This latter finding may point out the path to some very remarkable discoveries on the basis of some psychiatric states, such as schizophrenia, for example.

Conclusion.

In conclusion, it may be noted that organic lesions are likely causally related to the mental disease observed in:

(i) *Chronic Infections*—syphilis, tuberculosis, disseminated sclerosis, presenile degeneration.

(ii) *Acute Infections* — encephalitis lethargica, erysipelas, influenzal complications.

(iii) *Severe Trauma*—severe blow on head, brain tumor, surgical removal or part of cerebrum.

(iv) *Less Severe Trauma*—subdural haehorrhage.

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Recently delivered papers by Dr. K. G. McKenzie, Dr. C. B. Farrar and Dr. A. W. Keith.

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RECENT ADVANCES IN THE HISTOLOGY OF THE PULMONARY ALVEOLI

M. H. BOOK
Department of Anatomy, University of Toronto

Accurate knowledge of the structure of the respiratory alveoli is obviously of the utmost importance, for it is in these units that the exchange of gases between the air and the blood takes place. The past decade has witnessed a revival of interest in the histology of the alveoli, with the result that our ideas regarding this subject have undergone considerable modification. It is interesting to note that the latest conception of the structure of the lungs coincides wih the views advanced almost at the beginning of the microscopic era in anatomy, views, however, which were in eclipse for many years until recent studies served to substantiate them in great measure.

The Classical Description of the Alveoli

Until a few years ago the architecture of the alveolar walls was considered to be definitely settled and accepted by the great majority of observers. It is true there were one or two dissenting voices, (for instance, that of Miller, to be discussed below) but since their ideas were really only modifications of the classical description they received comparatively little attention. Thus most text-books described the alveolar walls as being lined with an uninterrupted layer of epithelial cells which covered the capillaries completely and were continuous with the epithelium of the bronchial tree. The epithelial cells were said to be of two distinct types—relatively small nucleated cells with granular cytoplasm, (called by some "septal cells") and quite large, flattened non-nucleated protoplasmic plates. A typical alveolar wall, then, consisted of a network of capillaries covered completely on both sides with epithelium. The septal cells were described as usually lying in the meshes of the vascular network, either singly or in small groups, and the remainder of the lining epithelium consisted of the non-nucleated squames. Reinforcement was provided by two separate networks of fibres, one elastic and the other reticular, while the interstices were filled in with a small amount of intercellular fluid.

The above "classical" description is usually associated with the name of Kölliker (1899), although he was only one of a number of workers, including Elenz (1864) and Eberth (1864) to study this problem. Many of the very first investigators (e. g. Rainey, 1855, Zenker, 1861, Munk, 1862) denied the presence of an epithelium covering the capillaries and thought that the latter were exposed naked to the alveolar air. Their methods of investigation were considered crude, however, and when von Recklinghausen in 1862 introduced his silver impregnation method for demonstrating cell outlines this procedure was at once adopted in the study of the lungs. By inflating the lungs through the trachea with a weak solution of silver nitrate and exposing the tissue to light certain irregular lines resulted which were interpreted as the outlines of the two types of cells described above, constituting a continuous respiratory epithelium. This, it was felt, was conclusive evidence and for some 60 years this view was accepted without question, and nearly all the textbooks in that period presented the same version of the structure of the lungs.

Recent Researches

Of late years this theory has been attacked by a number of workers who have been directing their criticism at one vunerable point. The only method which reveals the non-nucleated plaques is the silver-impregnation technique, and, although there is no question about its reliability in certain tissues, such as mesothelium and endothelium, there is grave doubt about the interpretation of the lines in the pulmonary alveoli. Any one who examines silver preparations of the lungs with an open mind is struck by the fact that the so-called cell outlines are quite irregular in thickness and continuity, enclose spaces of greatly varying area, and often end abruptly for no accountable reason. The picture is in distinct contrast to the regular cell outlines of a piece of silver-impregnated mesentery, for instance. Several authors have definitely denied that the irregular lines are cell outlines. Bloom in Maximow and Bloom's text-book (1931) suggests that they are imperfectly impregnated reticular fibres. Josselyn (1935) thinks that many of these lines outline the profiles of capillaries. Seemann (1929) concludes his article by saying that "the question of the nature of the silver lines between the alveolar epithelium remains open. Certainly they have no connection with either the capillary borders or reticular fibres." Obviously the nature of these lines is at present not settled; there is, however, a rapidly growing body of opinion that refuses to accept the view that they are cell-outlines.

This new interpretation of the results of the silver-impregnation technique disposes of the main prop supporting the classical theory of the respiratory epithelium. There is more evidence to disprove the presence of the non-nucleated plates. They have never been seen to play a part in any sort of inflammatory process, nor have they been found in sputum. They have never been described as giving rise to tumors. Finally, Lange 1909) stained the washings from the lungs of animals, and although he discovered numerous other types of cells he was unable to demonstrate the non-nucleated squames—a striking finding when one recollects that according to the old theory they form the largest portion of the covering of the alveolar wall and so should be very numerous.

The acceptance of the presence of the plates has become so ingrained in the minds of some uncritical observers that they have gone to great extremes to "prove" their occurrence. One worker felt that the reason they could not be stained in scrapings or washings of the lungs was their extreme thinness. Schulha (1933) proposed a compromise by suggesting that the large plates were really flat flanges projecting from the sides of the nucleated cells. Another author carried this idea a step further by saying that these large flanges were broad and thin as long as the cells were attached to the alveolar walls and thus subjected to the stretching of the lungs, but when separated from their attachment and the tension relieved they became rounded, nucleated cells. One has the feeling that these individuals are fighting for a dying cause.

Josselyn (1935) examined sections of rabbit lungs from which the blood had been washed and was able to identify only two types of cells—the endothelial cells of the alveolar capillaries, which formed by far the greater number, and an occasional cuboidal cell with a round nucleus and deeply staining, granular cytoplasm—the septal cells. Loosli (1935) varied the procedure somewhat by collapsing the lungs by means of phrenicotomy and pneumothorax and made similar observations. Both workers carried out extensive studies of the cells of the alveoli, using various fixatives and staining methods, and are emphatic in denying the presence of the non-nucleated plaques. Their papers are typical of a number of others which present the same conclusions.

If there are no non-nucleated plates in the alveolar lining then it is apparent that the latter must be a discontinuous membrane and that the capillaries are uncovered. There is, therefore, between the blood in the capillaries and the alveolar air only the endothelium and a varying, small amount of tissue fluid. (Obviously the capillary walls could not function if they were quite dry). Theoretically, this arrangement is the most efficient for the rapid transference of gases, for it interposes between the blood and the air the irreducible minimum of a moistened semi-permeable membrane. This conception helps to explain the rapidity of action of inhalation anaesthetics. As a means of administering drugs for very rapid absorption the lungs are not far behind the intravenous route. Policard has compared the respiratory surface to a vast open wound. When one recalls that the surface area of one lung on ordinary deep inspiration has been conservatively estimated by Willson (1922) to be 70 square metres, the rapidity of absorption in the lung becomes easier to understand. The lung is the only organ in the body in which the capillaries are in actual contact with the exterior.

If the Kölliker description of the structure of the alveoli has been rejected it must not be thought that there is now unanimity of opinion regarding the septal cells. Of the recent writers who believe that the alveolar lining is discontinuous, one school affirms that the septal cells are of entodermal origin (Aschoff, Seemann, Carlton), while another claims that they are derived from mesenchyme and are in fact histiocytes (Policard, Lange, Fried). Most of the evidence seems to favour the first group. An interesting possibility is that the septal cells really include more than one type of cell; this aspect awaits further study and improved staining methods.

Mention should be made at this point of another view of the alveolar lining, that of W. S. Miller (1932). This worker claims that the lining is continuous and made up of a single type of flattened, nucleated cells of entodermal origin. His studies are based largely on the examination of pathological material, pneumonic lungs for instance, in which oedema fluid has detached a lining membrane from the alveolar wall. Most observers feel that Miller's epithelium is nothing more than an inflammatory exudate made up of serum, fibrin, etc. which has shrunk away from the underlying tissues through action of the fixative.

The Alveolar Phagoctyes

The origin of the large mononuclear phagocytes of the lung has provoked a great deal of discussion. These cells, which play such an important role in clearing the pulmonary alveoli of foreign particulate matter, are known by a variety of names. As "dust cells" they are found normally in the lungs of town-dwellers, ingesting the dust and carbon brought to the alveoli in the inspired air. The are the "heart failure cells" which phagocytose the extravasated red cells in chronic passive congestion of the lungs. They also form an important line of defense against bacteria which have reached the interior of the lungs by means of the air-passages. Experimentally, they are easily demonstrated by introducing finely-divided foreign matter into the trachea—Indian ink, milk, bacteria, etc.

An exhaustive review of the vast literature concerning these cells is not feasible in this paper. Briefly one may summarize the main theories as to the derivation of the alveolar phagocytes as follows:

(1) The endothelium of the alveolar capillaries.

(2) The monocytes of the blood.

(3) The septal cells previously described, assuming them to be of entodermal origin.

(4) The same septal cells which are, however, presumed to be of mesenchymal origin. (Histiocytes).

(5) Multiple origin; more than one of the above sources.

The first theory is now of little more than historical interest for even some of its own proponents have abandoned it. For instance, Foot (1927), one of the earlier champions, has definitely denied it in favour of the second view. Metchnikoff was one of the first students of the defense mechanism of the lungs; he maintained that "the large 'dust cells' of the respiratory channels are . . . . in reality . . . . white corpuscles that have immigrated into the alveoli and bronchi." This theory fell into disfavour for a time but of recent years has been revived in some quarters. If the white blood cells do give rise to alveolar phagocytes they are certainly not the only source, as Westhues (1926) proved in an interesting piece of work. A normal rabbit was perfused with saline after which an intratracheal injection of India ink was made. A piece of lung was then excised and incubated in saline for half an hour while a control fragment was removed and immediately fixed. Microscopic examination showed that while phagocytosis was absent in the latter it was active in the incubated fragment. This simple experiment thus indicated that the dust cells could only have had their origin in the lung.

The majority of investigators incline to the belief that these phagocytes are derived from the nucleated elements in the alveolar walls—the septal cells. As has been pointed out above, opinion is sharply divided as to the nature of these septal cells. One group of workers claim that they are the same cells which in the embryo formed the continuous lining of cuboidal cells of the respiratory alveoli, and hence are of entodermal derivation. Their opponents assert that they are of mesenchymal origin, for only cells derived from mesenchyme (and particularly the histiocytic system) are actively phagocytic. This reasoning is faulty. The histiocytic (or reticulo-endothelial) system exhibits its markedly phagocytic properties characteristically when the foreign material enters the body directly (intravenously, subcutaneously, intraperitoneally, etc.) Under such circumstances the alveolar phagocytes, as Cappell (1929) has shown very clearly, are relatively inactive. It is only when the foreign material is brought into *direct contract* with the alveolar phagocytes, as in intra- tracheal administration, that thes cells exhibit their greatest activity. Moreover, Carleton (1934) and others have demonstrated that phagocytosis by epithelial tissues is also quite possible.

Although the matter is far from being settled the weight of the evidence at present available would seem to be in favour of the view that the alveolar phagocytes are derived from the septal cells, which are in turn of entodermal origin. It is quite possible that the circulating white cells also contribute, but to a much smaller extent.

Embryological Researches

An oft-quoted piece of evidence in favour of the "classical" conception of the alveolar lining has been the development of the mammalian lung. It was formerly stated that the foetal

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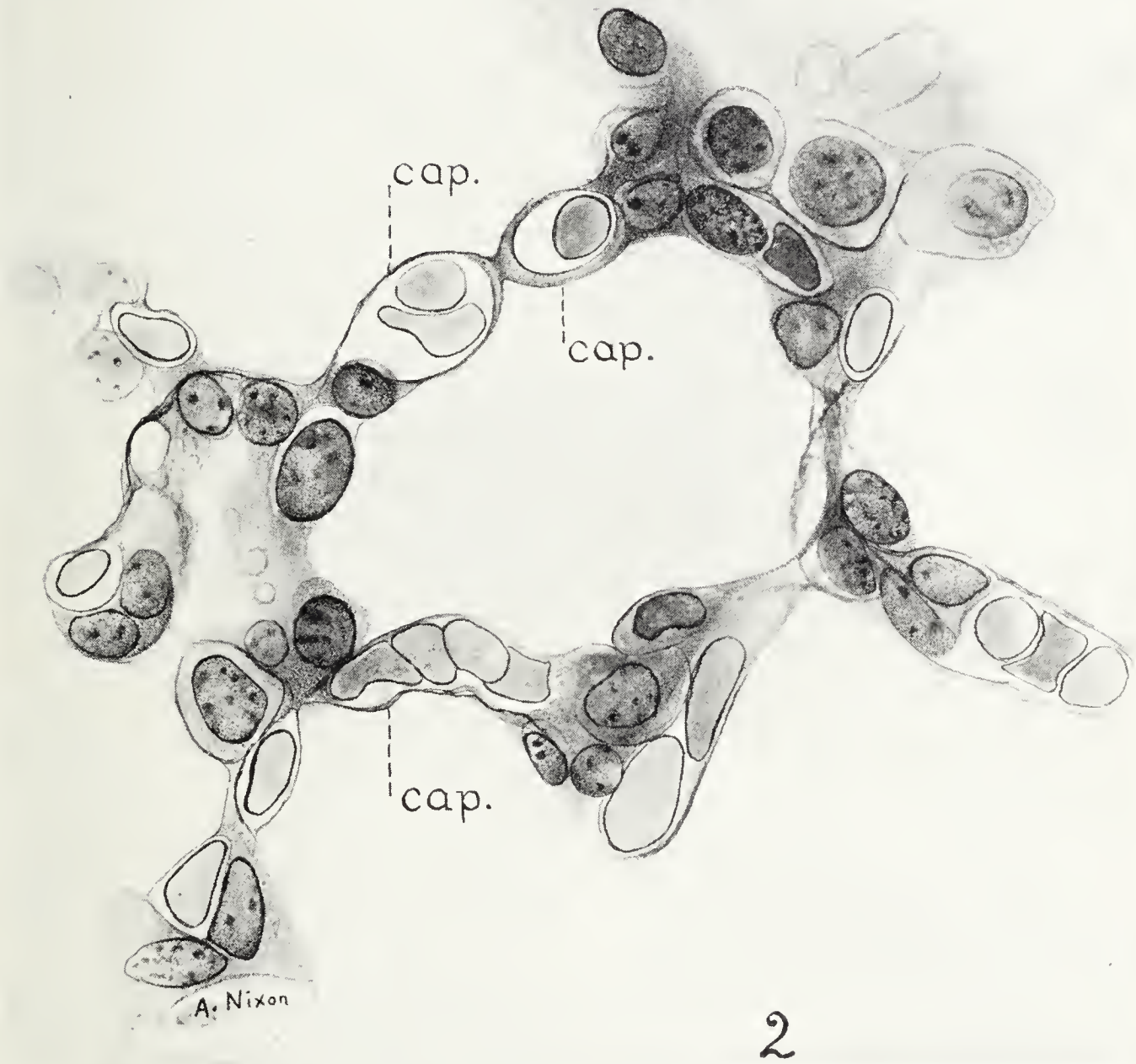
EXPLANATION OF FIGURES

No. 2. Newborn guinea pig lung. 4 mu section. Formol-Zenker fixation. Mallory's connective tissue stain. Single alveolus showing apparently naked capillaries in walls.

No. 3. Adult rabbit lung. Animal perfused with silver citrate solution. Lung fixed in 10% formol. 90 mu section. Mallory's connective tissue stain. Surface view of alveolar walls showing capillary network and alveolar pores (p).

These figures show clearly the importance of studying thick sections of lung in addition to the conventional thin sections.

(This plate is reproduced by kind permission of the Wistar Institute Press from Josselyn, L. E., 1935, the nature of the pulmonary alveolar lining. Anat. Rec. 62; 147).



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lung is lined by a cubodial epithelium which, with the first few respirations after birth, assumes the flattened shape characteristic of the fully developed adult lung. This description is absolutely erroneous and it is difficult to understand how it received such wide-spread credence when the facts are quite different and fairly easy to ascertain. In reality the development of the pulmonary alveoli is a continuous process and the birth of the foetus does not cause such marked changes in the epithelium as were formerly taught. The pulmonary buds are lined by a continuous sheet of epithelium till about the 5th month of intra-uterine life. At this period the lining becomes discontinuous; this is probably due in part to irregular respiratory moments attempted by the foetus. Some workers (e. g. Wenslaw, 1930) think that the epithelium becomes discontinuous because of the passive stretching of the membrane; studies that are being carried out at present in this laboratory tend to confirm this view. Others, however, (e.g. Alice, 1933) claim that the epithelium degenerates and disappears completely. In any case the capillaries, which were formerly separated from the lumen by the cuboidal epithelium, now project uncovered into the alveoli. Most likely it is this uncovering of the capillaries which determines the viability of a foetus born before term; it would seem that only when the vascular network is exposed in this way to the alveolar air is there sufficient possibility of the interchange of gases to enable the foetus to carry on its independent existence.

This expansion of the alveoli, alveolar sacs and alveolar ducts, with the coincident exposure of the capillaries, is quite advanced at the end of the gestation period. The foetus has been breathing irregularly for some weeks and the lungs are filled with fluid (some of it probably amniotic). Of course it does not drown because it is receiving oxygen through the placenta. After birth the fluid in the lungs is disposed of in several ways—some evaporates, some is coughed up, some is swallowed and probably a little is absorbed by the pulmonary circulation. At the same time the alveoli are opened up a little wider and the blood channels contain more blood. These are the principal changes in the lungs immediately after birth; the individual cells undergo no change that one can recognize under the microscope. There is certainly no flattening out of an hypothetical cuboidal alveolar lining. As is well known, th opening up of the lungs proceeds slowly and irregularly, and it is not till some days after birth (possibly 7 to 10) that all the alveoli are fully expanded.

The Alveolar Pores

If the origin of the alveolar phagocytes has been made more controversial by the latest work on the histology of the lungs the status of the alveolar pores, on the contrary, has become considerably clarified. These openings, (often called Kohn's pores) which are easily demonstrated in the lungs in lobar pneumonia because strands of fibrin are seen to extend from one alveolus to its neighbor through obvious gaps in the intervening wall, were considered by many workers to be present only in diseased lungs, after the epithelium had desquamated in part. Others (Ogawa, 1920, Joannides, 1931, Macklin, 1935) found them in the normal lungs of many mammals, including man. In the light of the recent researches these pores are easily explained—they are simply the interstices in the capillary network which forms the bulk of the alveolar wall. It is altogether likely that these vents are transitory, depending on the integrity of the thin film of tissue fluid which fills in the meshes of the capillary network, like water poured through a fine sieve. It has been suggested that the vents are variable in number and situation. For instance, fixing a lung for examination by inflating it through the

trachea is likely to show more of them than fixing it in a semi-collapsed condition, as is often done at autopsy; the inflation opens up the meshes of the capillary and fibrous networks. Again, a slightly oedematous lung would probably cause many of the pores to be sealed over with exudate. The accompanying illustration, taken from Josselyn's paper, shows the pores clearly. (Fig. 3.)

Summary.

One may summarize the latest researches on the microscopic anatomy of the lungs by stating that the alveolar wall is made up primarily of a close-meshed network of capillaries, supported by two other independent networks of reticular and elastic fibres. Scattered here and there in the wall are the septal cells—more or less cuboidal cells with spherical nuclei and granular cytoplasm. Filling in the spaces between these structures is a small amount of tissue fluid; the absence of this fluid from the meshes of the networks results in the alveolar pores.

The derivation of the septal cells is not clear, although most authorities think that it is from entoderm. Whatever the origin of the cells it seems fairly certain that, when properly stimulated by the presence of foreign material in the alveolar lumen, they give rise to most, if not all, the alveolar phagocytes.

Recent embryological studies have produced an entirely new description of the development of the pulmonary alveoli, confirming the above outline of the structure of the adult lung.

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THE SCHOOL-CHILD'S BREAKFAST

Many a child is scolded for dullness when he should be treated for undernourishment. In hundreds of homes a "continental" breakfast of a roll and coffee is the rule. If, day after day, a child breaks the night's fast of twelve hours on this scant fare, small wonder that he is listless, nervous, or stupid at school. A happy solution to the problem is Pablum, Mead's Cereal cooked and dried. Six times richer than fluid milk in calcium, ten times higher than spinach in iron, and abundant in vitamins B and G, Pablum furnishes protective factors especially needed by the school-child. The ease with which Pablum can be prepared enlists the mother's cooperation in serving a nutritious breakfast. This palatable cereal requires no further cooking and can be prepared simply by adding milk or water of any desired temperature. Its nutritional value is attested in studies of Crimm et al who found that tuberculous children receiving supplements of Pablum showed greater weight-gain, greater increase in hemoglobin, and higher serum-calcium values than a control group fed farina.

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THE SUBSCORBUTIC STATE

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In order to appreciate the nature and importance of the sub-scorbutic state it is necessary to discuss at some length the subject of acute or florid scurvy, all the more so since very important contributions have been made in that field within the past few years.

The discussion of acute scurvy is of great interest in itself. It tells the tale of one of the most clean-cut and satisfactory chapters in medical history. From the old days when the empirical discovery was made that "lime" juice* was specific for the disease and when the latter in its florid form was practically eliminated from the British Navy (whence British sailors for many years were nicknamed Limeys) there has been a steady and orderly development. This has been discussed extensively in most works on vitimins, perhaps most entertainingly by Still ('35), and we have here the intention of touching only the more important phases down to the most modern developments.

Etiology of Scurvy.

The rise of bacteriology had little effect on the theory of scurvy or its treatment though scurvy, like every other disease, was investigated for possible bacteriological causes. And the dawn of the vitamin theory in the early years of this century very quickly incorporated the excellent old studies of scurvy with the new discoveries. In 1912 Holst and Fröhlich definitely established scurvy as a deficiency disease, produced it experimentally in the guinea-pig and opened the door to its laboratory investigation.

Before this there had been two main causes of confusion in the study of the disease. For a time there was a distinction made between the adult form and a variant found in infants; the latter was carefuly and extensively studied by the famous Sir Thomas Barlow and consequently was known for a long time as Barlow's disease; it is curious that it should have been considered as a distinct clinical entity since Barlaw himself was very specific about its scorbutic character (1883 and 1894) but, especially in Germany, this distinction was so emphasized that as late as 1918 such authoritative writers as Aschoff & Koch could seriously discuss the question of whether these two diseases were the same. However, the question has long been settled. The other source of difficulty has been the tendency for more than one deficiency disease to occur in the same subject, especially in infants (Barlow loc. cit., Fröhlich and many others). Where the diet is deficient in one vitamin there is a great likelihood of its being poor in others also. Hence scurvy may be complicated by rickets, beri-beri, or both, especially when the custom of feeding infants patent foods of poor vitamin content was in vogue (Hess, pp. 248 et seq.) This difficulty has also been elucidated and scurvy is sharply demarcated from the other deficiency diseases, owing to the advance of experimental technique.

The most recent phase in the study of florid scurvy began with the identification by Tillmons and by Szent-Györgi simultaneously (1932) of hexuronic acid as the vitamin-C already studied by dietitians empirically. (Szent-Györgi had already isolated this compound in '28 but had not recognised it as the vitamin). Since then a series of studies (Szent-Györgi, Horst and Zilva, Reichstein and Oppenhauer, Ha-

*It may be worth pointing out that lime was formerly used synonymously with lemon. Actually true limes are very feeble antiscorbutics.

worth et al) have eliminated the possibility of the active principle's being an impurity associated with the acid, by transmuting the latter into an inactive compound and back again into the active form, and by actual synthesis of the acid.

Vitamin C. Requirement and Content of Foods.

A further great advance has been made in methods of assessing the concentration of the vitamin in any given food or tissue. The first methods consisted in feeding guinea-pigs (one of the few laboratory animals susceptible to typical scorbutoid changes) graded doses of the food in question and observing how much was necessary to keep the animal from contracting scurvy. This method had the double disadvantage of being very rough and very slow. About 1924 Höjer discovered that changes could be seen in the incisor teeth of the pigs at an early stage of the disease, thus cutting down the time-factor, and that these stages could be roughly graded according to the degree of change present in the teeth, thus allowing a certain increase in accuracy. But Göthlin (1931) devised a test of the strength of skin-capillaries (the capillary system being particularly susceptible to scorbutic changes) which allows us to detect the onset of scurvy in the living human, though it has not yet been confirmed that this test is specific and dependable; in any case it is certainly of importance. Key and Elphick (1931) described a method of staining the adrenal cortex with silver as an alternative to examining the teeth but excepting that this tissue is easier to prepare for the microscope the method does not seem to be an advance on observing the teeth, and other writers have found it only useful to within a wide approximation (e.g., Dann and Cowgill, 1935). However, Harris and Ray developed (1933) a method of direct chemical assay by titration with dichlorphenol-indophenol which allows accurate investigation for vitamin content of all substances, including those too scarce for feeding purposes or unsuitable for feeding.

All this is very gratifying, and it would appear at first glance as though scurvy were something with which North Americans had as little concern as with bubonic plague, leprosy or epidemic smallpox. Indeed this attitude is widespread in the lay population and appears to have some foodhold even in the medical profession. An instance of this is seen in an article printed recently in a popular magazine in which the author, an M.D., ventures to assert that the average diet is so mixed as to render superfluous any accessory dose of vitamins such as the daily glass of orange-juice; he confirms this statement only with the evidence that in a long practice in a large New York hospital he saw only one case of scurvy and that was due to eccentric diet.

In this statement he ignores some very important considerations. It is quite true that frank scurvy will probably be confined herceforth, as far as North America is concerned, to abnormay circumstances such as isolation, extreme poverty, dietary fads and ignorance. But to dismiss frank scurvy does not settle the question of vitamin C dietetics; to assume that it does so is rash and may be mischievous. Florid scurvy is the end-result of a long process, the final evidence of hidden changes. Writers (e.g., Park, '35) have taken the fact of its apparently sudden onset as evidence that there are few if any physical changes early in the course of vitamin-C deficiency. The present writer would take it rather as evidence that changes must have been taken place extensively. Nor do spectacular recoveries of apparent health, upon vitamin therapy's being applied, negative the fact that extensive changes must have taken place in at least the bony structures which must require considerable time to repair. Scurvy is not a brief, sudden disease but one requiring, in man, a latent period of from six to nine months.

Latent Scurvy.

Park himself contributes evidence of what he considers the earliest appreciable changes and remarks that there are possibly less significant prodromal changes. The skeleton is affected long before patent symptoms occur. In short, the latent period in scurvy is a period of pathological changes in the tissues and throughout this time the subject, while apparently healthy or suffering only from slight, ill-defined malaise or commonly disregarded minor symptoms, is really in a diseased state.

But what if this latent pathological state be kept under the surface by the administration of just enough vitamin C to prevent the outbreak of the frank form? This is easily possible, for whereas the daily dosage required to keep a man or a guinea-pig in perfect health is definitely determined by various tests, the administration of a lesser dose will not produce acute scurvy. There must be a graded series of conditions merging into one another from normal health to fatal scurvy. How are these conditions characterized? Will their effects be variants of the symptoms of frank scurvy or will the prolongation of the condition over many years produce results which are not indicated by the picture seen in the usual acute condition? These are questions to which only scattered and incomplete answers have been offered, the most striking of which is that by Rinehart, Connor and Mettier, discussed below. What evidence have we of endemic latent scurvy?

Aschoff and Koch in their monograph, Das Skorbut, describes in detail three stages in the development of scurvy, a preliminary, an acute and a final. In the first of these which should be most closely akin to latent scurvy we have a definite series of symptoms: fatigue, loss of appetite, dryness and muddiness of the skin, joint-ache, muscle-ache with a pronounced anxiety to remain seated rather than standing, headaches, bleeding gums, depression and heightened pulse-rate. With the possible exception of bleeding gums, these are all such non-specific symptoms and can be traced to so many causes that it would be absurd to attempt to attribute them to the scorbutic condition in more than a fraction of cases. Indeed one could not say with certainty that because they were bound at the beginning of acute scurvy they would be characteristic of a chronic, latent scurvy. Nevertheless, in a civilization where these symptoms are frequent indications of imperfect health widespread in the population and where a great percentage of the population still has a predominantly carbohydrate and denaturised diet, either through necessity or choice, it is a fair question to ask whether in some cases the symptoms are not, if only in part, due to a latent scurvy, more or less severe in degree.

Even with the best will in the world it is not always easy to establish an adequate intake of vitamin C unless an apparent excess is assured by a considerable margin. For example, really fresh fruit has far greater potency than old fruit, even when the latter is not dried or otherwise deteriorated (ibid. P. 175). Proper cooking has long been recognized as a necessity in the preservation of antiscorbutic value in foods; prolonged cooking, warming-tables such as are used in cheap restaurants, will destroy much of the value in foods. Bramwell, Hoyle & Zilva (1930) found that there was significant difference in antiscorbutic potency between different types of apples. This list might be protracted into a considerable paper in itself. The point is well enough established, however, that we are far from knowing definitely how much vitamin we are taking in any given quantity of food and that our error will almost always be on the side of crediting the food in question with too much. Vitamin C is a highly labile compound, apparently more so than any of the other vitamins, and it may elude us even in a diet supposedly adequate.

It has one other peculiarity which makes it necessary to watch it carefully in our diet. It cannot be stored in the body. Hess (p. 75) states that guinea-pigs which had lived for some time with an excess of vitamin C equalling double the necessary ration in the diet developed scurvy just as rapidly when put on a scorbutic diet as pigs which had been living on the bare minimum; the excess had not been stored. Therefore we cannot carry over a reserve of antiscorputic vitamin from seasons when foods containing it are cheap and palatable (summer and fall)* to seasons when it is hard to get, expensive, and impoverished in quantity even in the foods where it is found most commonly.

Thus, our popular article-writer to the contrary notwithstanding, it is not so certain that everybody gets all the vitamin C they could use to advantage. Göthlin (1931) estimated that the human requirement was about 0.7 to 1.0 cc. of orange-juice (a very constant and highly potent source of the vitamin) per kilogram per diem. A small orange gives not more than 50 cc., and it is doubtful if the average men takes enough fresh milk, salad vegetables, or *properly-cooked* greens and potatoes to much more than cover the remainder of his requirement—20 cc. for a 70 kg. man. What then if he does not get that daily orange? Even with smaller people there is evidence to show that some require more than others and the error should be on the right side.

There is a certain shyness up among sensible people against food fads. But the morning orange-juice is not a fad. Our "normal" diet is not normal at all, for our race; presumably our remote ancestors were largely fruit-eating; certainly our more recent predecessors were mainly inhabitants of the warmer regions where fresh fruits and dairy-products from animals pasture-fed all year were staples. Therefore our systems must have been developed to demand vitamin C in considerable quantities. If our intelligence permits us to live in colder climates by artificially reproducing sub-tropical conditions of temperature in our homes and under our clothes, it must also maintain a sub-tropical diet. Of course, as mentioned above, individuals probably vary greatly (if guinea-pigs are criteria, we certainly do) and a process of natural selection might eventually produce a race able to subsist normally on a reduced vitamin C ration!

Enough has been said to support the claim that latent scurvy is probably endemic in our population and that it may possibly account for many minor ailments and some lowered resistance to infection. In this latter field Robertson ('34) has made an extensive review covering sixty-five papers, which almost unanimously confirm the view that a deficiency in vitamin C, even though not total, lowers resistance to infections of many sorts and to other toxic invasions.

It remains to discuss the possible relationship of vitamin C inadequacy to more serious disorders. Before doing this we will find it useful to examine the theory of how vitamin C participates in metabolism and how its lack affects the tissues in general—in short, the basic theory of scurvy.

Pathological Process in Scurvy.

This question itself has still not been fully answered. The prevailing view, however, is briefly as follows: A lack of vitamin C in the diet retards or entirely prevents the formation of intercellular substance by cells of the connective-tissue group. Woolbach and Howe ('33) show that bones will not knit in a scorbutic subject and that a saw-cut in a scorbutic bone will not heal; but when vitamin C is supplied to such a subject the healing takes place

*While this is generally the distribution Park et al, in the paper already quoted, found the relation inverted, which was explained by their subjects being infants dependant on orange-juice for their vitamin and by the fact that oranges were cheaper in that district in winter than in summer.

with abnormal rapidity; they take this to indicate that the substance necessary for healing have been mobilised all but one factor without which they are unable to "set" and this factor is evidently vitamin C.

Another supporting piece of evidence for this theory is supplied by Park et al in the paper cited above. In the normal growing bone the cartilage of the epiphyseal plate is continually porliferating so that it would eventually become of considerable thickness; however, the margin towards the shaft of the bone is continually being eaten away and replaced through a series of processes — first becoming calcified with death of the cells, and then being replaced by bone trabeculae—so that it remains a very narrow zone. If, however, normal proliferation of bone is inhibited while the cartilage continues to proliferate, the zone of calcified cartilage matrix with dead cells will grow progessively wider. This is the case in latent scurvy; bone-growth is inhibited while cartilage, less susceptible to mild scurvy, does continue to grow and die and form a wide band of dead matrix heavily charged with calcium and seen as a dark shadow (the scorbutic lattice) on an X-ray *proof*.

These changes in the skeletal structures are early and typical. An equally constant and even more serious effect of florid scurvy is seen in the blood-vessels. Sub-dermal hemorrhages are almost characteristic of the acute disease, their location being generally determined by differential variance (even if slight) of pressure or insult; the first hemorrhage is frequently found after a light blow; the commonest location is in the legs, where the venous pressure is greater (Aschoff & Koch, 1918). This bleeding is not accompanied by demonstrable rupture of the vessels and has no obvious relationship to thrombosis in the smaller vessels (Ibid); consequently it is assumed to be due to diapedesis (if the term may be properly applied to erythrocytes) through the walls of capillaries which have been weakened by loss of cement-substance or (as it has occurred to me) by loss of support from surrounding fibrous tissue. These hemorrhages are intermittent as is shown by the presence of deposits of blood pigment in association with fresh extravasations and are by no means confined to the skin but may occur in any part of the body where stress is placed on the capillaries, notably the gums. There is also a tendency to edema especially in the great body-cavities (Aschoff & Koch) and in the legs (Hess, p. 196) the cause being doubtless similar to that of the bleeding.

This is the fundamental background of scurvy as far as we know it. Let us now suppose that we have such a condition not in a sudden and acute degree but chronically and not severely enough to cause any gross pathological symptoms such as are characteristic of acute scurvy. First we must set aside all infectious conditions possibly due to lowered resistance, as noted above. It is the opinion of Aschoff & Koch and many others that the striking and gruesome involvement of the gums so often seen is not a primary effect of scurvy but due to lowered resistance, the constant exposure of the tissue to the violent strains of mastication and the bacterial flora constantly present in the mouth. This is borne out by the fact that antiseptic treatment will frequently ameliorate this symptom in the absence of vitamin therapy, as was shown in the work of that able pioneer on scurvy, Lind (1752). One would be tempted to relate the frequency of "pyorrhea" to latent scurvy, and Robertson quotes authorities to support this, but that is beside the point. In guinea-pigs the presence of scurvy certainly predisposes the animals to various infections, as I have had occasion to learn, and there can be no resonable doubt that this is also the case in man.

It is interesting to note that many organs supposedly of importance in resistance to disease, notably the elements in bone marrow, are closely related to

the connective tissues already known to be involved and are known to be affected by scurvy, although, how they are affected has not been determined. (Höjen, '24; Findlay, '23).

Such are secondary effects. But are there no primary effects due to the long weakening of the tissues and impaired metabolism? It must be remembered that the group of tissues chiefly affected by scurvy are very active. Bone is in a constant state of flux; the writer (1936) has had occasion to point out that articular cartilage is subject to continual wear which it must replace; teeth while apparently incapable of regeneration are sensitive to inadequate diet. While the animal is young tissues are growing and a deficiency in any essential is bound to have a deleterious effect; and even in the adult state there must be replacement of wear and maintenance of normal metabolism. Autopsy on cases of acute scurvy shows that the ventricles of the heart are frequently not only distended but that actual hypertrophy has taken place. place. Cancellous epiphyseal bone is resorbed, leaving a mere web of fibrous tissue. marrow - framework (Gerüstmark).

Possible Relation of Subscorbutic State to Rheumatoid Arthritis.

All these things are very suggestive and offer a promising field for research. During the past two or three years Rinehart, Connor and Mettier (34 a and b, '33) have been working on the possibility of vitamin C deficiency being one cause of arthritis; giving guinea-pigs a diet deficient in but not totally lacking in vitamin C, they obtained results in the joints not unlike those of "rheumatic fever"; when they superimposed a mild systemic infection on the condition the resemblance was increased.

The present is the time to continue such work. On the one hand the demonstration of a relationship between latent scurvy and any common pathological state would stimulate further research and popular education and help to abolish the complacent attitude instanced above. On the other hand it would forestall this very improvement in diet which, one hopes, will come eventually in any case, and would enable us to make a study of all the effects of vitamin deficiency before material is no longer available. For though a simple raising of dietary standards would wipe out these effects without the intervention of research it is needless to emphasize the importance of knowing exactly what we have wiped out.

To the academic research-man the study of vitamin metabolism will doubtless offer a valuable key to the secrets of vital processes and from knowledge so gained may come further reactions on medical science. But we to-day have within our grasp a task difficult and important enough to tax all our powers, in confirming and completing our knowledge of the vitamins, and especially vitamin C, in our every-day diet and health.

Bibliography on Request to Author.

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A COMPARATIVE ANATOMY OF THE CIRCULATORY SYSTEM

JAMES G. WATT, '40

The circulatory system has two main functions. The first is to carry food and oxygen from the intestine and respiratory surfaces to the tissues and organs of the body where these substances are needed, and to remove waste matter from them. The second is to conduct peculiar stimulating substances, which are made by every activity in the body, from the place of formation of the substances to the organs or cells which require these activators.

The media in which the above substances are carried are the blood and lymph. In lower forms of animals the blood and lymph are conducted through the body in the same vessels. This is a haemo-lymphatic system. The mammal however, possesses separate vascular and lymphatic systems. The latter is directly connected to the veins of the mammal.

The Circulatory System of the Earthworm

The blood vascular system of the earthworm consists of several vessels. There is no real structural difference to distinguish them into arteries and veins.

A large dorsal vessel runs the whole length of the body, from the posterior end to the pharynx. It is contractile, and the blood is driven forwards in it. This vessel supplies blood to, and receives blod from the gut. Many small vessels from it, metamerically arranged, communicate with the intestine. It sends two large vessels to the oesophagus in the tenth segment. In segments 7-11 five pairs of pseudo-hearts are derived from it. These are not true hearts but are large contractile vessels. They encircle the oesophagus and join a ventral subintestinal vessel, which hangs by a mesentary below the alimentary canal. The blood flows downward through the hearts and thence into the ventral vessel. This vessel sends metamerically arranged branches to the nephridia, and to the skin, which is the oxygenating surface.

The blood is purified by these organs, and is returned to the dorsal vessel by various paths. There are two lateral neural and one sub-neural blood vessels, in which the blood flows backwards. In each segment a pair of parietal vessels connect the sub-neural with the dorsal vessel, in the intestinal region.

The ends of the vessels are joined by capillaries. This makes a closed system. The blood consists of a fluid called plasma in which a red pigment, haemoglobin, is dissolved. There are a small number of white blood cells.

The earthworm's circulatory system is described because it is a good example of the metameric arrangement of blood vessels. Such a condition occurs in the mammalian embryo, but numerous anastomoses result in the union of many small vessels, so that in the adult mammal this condition is not so evident. Metameric arrangement is seen however in the case of the blood vessels of the vertebral column, and thoracic and abdominal walls.

The Circulatory System of the Crayfish

The crayfish has a single heart which is roughly hexagonal in shape. It consists of a single chamber which has thick muscular walls. The heart is situated in a cavity called the pericardial sinus, above the gut, and just below the cardiac region of the carapace. There are six fibrous bands attaching the heart to the walls of the sinus.

Three pairs of valved openings or

ostia admit blood from the sinus into the heart. Three arteries arise from the front of the heart and run forwards to supply the proventriculus and antennae. The two lateral ones are the antennary arteries. They also have gastric branches. Ventral to the antennary arteries are two hepatic arteries which run to the liver. At the posterior angle of the heart is a large vessel, which almost immediately divides into a dorsal abdominal artery to the intestine, and a sternal artery. The latter passes downwards through an opening in the ventral nerve cord, and divides into the ventral abdominal and ventral thoracic arteries. These supply the numerous limbs.

The vessels of the crayfish end in sinuses, not capillaries. It is an open circulation. The blood from all parts of the body is collected in the sternal sinuses. From here it is distributed to the gills by afferent branchial sinuses. The efferent branchial sinuses return the blood to the pericardial sinus where it enters the heart through the ostia. This pericardial sinus is part of the coelomic cavity.

The blood is a clear fluid containing white corpuscles, and it readily clots. It contains haemocyanin but no haemoglobin.

The crayfish portrays the simplest form of a true heart, that which contains only one chamber and has few valves. There are no veins.

The Circulatory System of the Clam

The heart of the clam consists of three chambers, two thin-walled atria, and one muscular ventricle between them. The ventricle is a wide contractile part of the dorsal blood vessel. This vessel runs forward in the body as the anterior aorta. A peculiar fact is that the pericardial sac and the ventricle are traversed by the rectum, and form a jacket about this part of the intestine. The anterior aorta passes into the spaces between the organs. A posterior aorta passes downwards and backwards from the posterior portion of the heart. Both these terminate in sinuses.

Below the pericardium and between the kidneys there is a vena cava, which gathers the blood from the foot and viscera, and conducts it outward to the gills and mantle. Two vessels return the blood from here to the atria.

The blood is colourless and contains white corpuscles.

The facts of importance concerning the vascular system of the clam are; that the heart and main blood vessels are dorsal to the intestine, that the blood vessels terminate in sinuses, and that there is no real structural difference between the arteries and the veins. Of primary importance is the fact that the heart alone is an advance over the corresponding structures of the earthworm and crayfish. It shows the tendency toward a complex heart with many chambers, which is characteristic of all higher forms of animals.

The Circulatory System of the Fish

The heart of a fish is situated in the floor of the pharyngeal region. It lies in the pericardium between the posterior gill clefts of each side. It has muscular walls and consists of four chambers which form an S. The posterior chamber is the thin-walled triangular sinus venosus. In front of this is the thicker walled atrium which is triangular in shape, with apex pointed forwards. The atrium is a single chamber. The ventricle is posterior and ventral to the atrium. It is very thick walled and muscular. From it, a fourth chamber, the conus arteriosus, passes forward through the anterior wall of the pericardium, where it becomes the ventral aorta. The heart has been looked upon as a thickening and twisting of the ventral aorta. The heart contracts from behind forwards in four beats, and there are valves situated at the openings between the chambers to prevent backflow.

The heart contains only venous, impure blood. This is driven forward in-

to the ventral aorta. This vesesl divides into several pairs of afferent branchial arteries. These corespond to the aortic arches in higher vertebrates, and are of the same number as the functional gill clefts. There are six pairs in Notidanid sharks, five in other Elasmobranchs (Skate), and four in Teleosts (Perch) and Ganoids (Sturgeon). The artery of the first branchial arch divides to supply also the hyoid arches. The afferent branchial arteries divide into capillaries in the gills, where the blood is oxygenated. Efferent branchial arteries collect the blood. Thus a complete loop is formed around each of the gill clefts by these. The loops are joined anteriorily and posteriorily by horizontal vessels. The last cleft has an efferent vessel on one side only.

Epibranchial arteries arise from the dorsal end of each complete loop, and run posteriorily to join the median dorsal aorta.

The anterior end of the ventral aorta divides into two external carotid arteries to the head. These are connected by a transverse vessel to the internal carotids, which arise from the anterior ends of the epibranchial arteries. These external carotids do not correspond to those of the frog.

The dorsal aorta terminates in front by sending small branches to join the carotid roots. Just before the dorsal aorta is joined by the epibranchial arteries it gives off a pair of subclavian arteries to the fore-fins. The dorsal aorta carries the purified blood to the body. Behind the pharynx it runs backwards along the whole length of the body, below the backbone. It becomes the caudal artery in the tail. The most important branches from it are; the coeliac to the stomach, and its hepatic branch to the liver; the anterior mesenteric to the intestine and its genital branch; the lienogastric and posterior mesenteric to the alimentary canal; and several paired branches to the kidneys.

The sinus venosus receives the whole of the blood returning to the heart by several very large veins called sinuses. These are not body cavities like the sinuses of the crayfish, but are enlarged parts of the veins. Two large hepatic sinuses return the blood direct from the liver to the sinus venosus. The rest of the blood is returned by two large ducts of Cuvier, which collect the blood from the various other sinuses of the body, of which there are thirteen. The caudal vein from the tail divides into two renal portal veins to the kidneys. An hepatic portal vein carries blood from the alimentary canal and spleen to the liver. The two portal systems correspond to the portal systems of the same name in the frog.

In the blood are red corpuscles containing haemoglobin. There are also white corpuscles and lymph. The fluid portion of the blood is called plasma and, in the elasmobranchs, is rich in urea.

The fish represents one of the lower forms of vertebrates, and several features of its circulatory system are comparable to the embryonic circulation in higher vertebrates. The mammal during its development has an S-shaped heart and a ventral aorta. The aortic arch is derived from a branchial artery similar to those in the fish.

The central position of the heart is also characteristic of vertebrates, but the position of ventricle and atrium in the fish is the reverse of that in higher vertebrates.

Although the frog has a renal portal and a hepatic portal system similar to that in the fish, the mammal's renal portal system only appears early in embryonic development, and disappears with the degeneration of the mesonephric kidneys.

The heart of the fish contains only impure blood, whereas that of higher vertebrates contains both pure and impure blood.

The circulation of the fish is closed and contains only a single circuit, that is, the blood from the respiratory organs goes direct to the body without returning to the heart in the interval.

The Circulatory System of the Frog.

The heart of the frog is a hollow, conical, muscular organ which lies with its apex backwards in the pericardial cavity between the oesophagus and the sternum. The pericardial cavity is a part of the body cavity, and it is lined by a thin membrane, the pericardium. There are five chambers in the heart. The first is the large conical ventricle. The second, the truncus arteriosus lies on the right central surface of this. It is smaller, more tubular and is incompletely divided by a spiral valve. The short distal part of this structure is the ventral aorta. It is separated from the part nearest the ventricle, which is longer and is called the conus arteriosus, by a row of semilunar valves. Three more semilular valves guard the opening between the ventricle and the conus.

There are two atria in the heart, a right and a left. They are thin walled chambers. The right is larger than the left and they are separated by a septum. They lie in front of the ventricle. There are openings from each into the ventricle, which are guarded by valves which prevent blood running back into the atria after they have ceased to contract.

Three arteries arise from the ventral aorta and conus arteriosus on each side. It is the binding together of these vessels for a short distance, which forms the truncus arteriosus. The three vessels of a side form the pulmocutaneous arch to the lung and skin; the common carotid going to the head region, and dividing into an internal carotid artery which passes lateral and dorsal to the eye and brain, and which contains a round swelling the carotid gland, and an external carotid artery. They also form a third vessel which is the aortic arch. It originates from the fourth branchial arch.

The two aortic arches unite posteriorily to form the large dorsal aorta. The latter before joining the aortic arch gives off a large artery, the coeliaco-mesenteric. This divides into a coeliac to the stomach and liver and an anterior mesenteric to the bowel and spleen. The dorsal aorta runs backwards immediately below the spinal column and gives off several branches. First are the paired renal arteries to the kidneys. The other abdominal branches are ovarian or spermatic arteries to the reproductive organs, and a small posterior mesenteric artery to the intestine. The dorsal aorta finally divides into two iliac arteries to the abdominal muscles and hind limbs.

The blood is returned to the heart by right and left pulmonary veins through a short common pulmonary vein. This enters the left auricle. The sinus venosus collects the blood from all the rest of the body by several large veins. They are the right and left precaval veins and a single postcaval vein. Each precaval comes from the head region and is formed by an external jugular which arises in the lower jaw, an innominate which is made by the junction of an internal jugular from the head and a subscapular from the shoulder, and a subclavian which receives the brachial from the arms and the musculo-cutaneous.

The postcaval vein arises by the junction of several renal veins with the veins from the reproductive organs. Just before it enters the sinus venosus it is joined by the hepatic veins from the liver. The blood returns from the legs by a femoral vein on the outside, and a sciatic vein on the inside of each limb. The femoral becomes the renal-portal to the kidney. The sciatic vein joins the renal-portal before the latter enters the kidney, where it divides into capillaries. These capillaries with those of the renal artery are collected to form the renal vein. Much of the blood then, has gone through two sets of capillaries, one in the leg, the other in the kidney. The arrangement whereby the blood passes through two sets of capillaries is called a portal system. The hepatic-portal vein collects the blood from the stomach, intestine, pan-

creas and spleen. It divides into capillaries in the liver. The hepatic vein conducts the blood from here to the sinus venosus. This is the hepatic-portal system.

The course of the blood is as follows: Venous blood goes from the sinus into the right auricle, and thence to the single ventricle. From here it travels by means of the pulmo-cutaneous and pulmonary arteries to the oxygenating surfaces where it is purified. It is returned to the left atrium by the pulmonary vein, whence it is driven into the ventricle.

The two atria contract at the same time so impure blood is forced into the ventricle with this pure blood. Muscular ridges or trabeculae prevent them from being mixed. On contraction of the ventricle, the impure blood is forced out first since it lies nearest the conus. It takes the most direct course through the pulmonary arteries. The pure and impure blood, which have mixed partially, pass out next. These take the next easiest course through the aortic arches. Pure arterial blood leaves last, and goes only to the carotids, where the great pressure, on account of the small size of the vessels, and the carotid glands, offers resistance to all but the oxygenated blood.

The frog represents the beginnings of a double circulation. The blood does not go to the body after passing over the respiratory surfaces but returns to the heart. The heart contains both pure and impure blood, and the trabeculae partly divide the ventricle into two chamber as an attempt to separate them. Since there are not two ventricles completely separated by a septum, as in the mammal, the frog must use various pressure devices to ensure the directions taken by pure and impure blood. The mammal accomplishes this by having two complete circulations.

The frog has an extensive lymphatic system. There are large body spaces into which the lymph capillaries empty. These are connected with each other and with the veins. The two veins into which they empty are the external jugular and the iliac. There are lymph hearts connected with the openings. These can enlarge and contract, and maintain the pressure in the lymph vessels.

The Circulatory System of the Bird.

The pigeon well exemplifies the circulatory system of birds. The pigeon's heart has four chambers, two atria, and two ventricles. There is no conus arteriosus or sinus venosus as in the frog. A septum completely separates the two sides of the heart internally, and as a result blood passes twice through the heart, once on the venous side, and once on the arterial side, in making a complete circuit of the body. Thus there is a complete double circulation.

Venous blood enters the right atrium, then passes to the right ventricle, by which it is forced to the lungs through the pulmonary artery. It returns to the left atrium by the pulmonary vein, from there it passes into the left ventricle, and thence to the body by the systemic trunk. There is a single aortic arch leading away from the left ventricle. The opening between the two is guarded by three semilunar valves. The aortic arch bends to the right side, and gives off at its apex the right and left innominate arteries. A carotid and a subclavian artery arise from each of these. The aortic arch, posteriorly, becomes the dorsal aorta. This has the same branches as the corresponding artery in the frog.

There are three venae cavae as in the frog. Each superior vena cava is formed by the union of a jugular, a brachial, and a pectoral vein. The inferior vena cava arises from the junction of the two iliac veins in front of the kidney. Each iliac vein is formed by a femoral, a renal and a hypogastric vein. The latter passes upward through the kidney. A few small branches from the femorals to the kidneys are considered to represent a renal portal system. The epigastric vein which takes blood from the greater

omentum represents the anterior abdominal vein of the frog.

The lymphatic system consists of paired main vessels, the thoracic ducts. These are united posteriorly. There are two non-contractile pelvic lymph hearts.

The Circulatory System of the Mammal.

The rabbit well portrays the typical mammalian circulation. The heart of the rabbit lies in the front part of the chest, and is enclosed in a thin membranous pericardium. There are two ventricles and two atria. The sinus venosus is merged in the right atrium, and there is no conus arteriosus.

Three venae cavae corresponding to those in the frog open directly into the right atrium, and two pulmonary viens open into the left atrium. Each superior vena cava is formed by the union of a subclavian vein from the limb with an internal and an external jugular vein from the head region. The right superior vena cava also receives the azygos vein from the chest wall.

The inferior vena cava is a large medium vessel lying beside the dorsal aorta. It is formed by a pair of internal iliac veins from the back of the thighs, and a pair of external iliac veins from the inside of the thighs. It is also composed of the iliolumbar veins from the abdominal wall, and the genital, renal and hepatic veins.

The renal portal system disappears with the degeneration of the mesonephric kidneys. The blood from the stomach, pancreas, spleen, and intestine is conducted to the liver by the portal vein, so there is an hepatic portal system.

The persistent left aortic arch forms the only connection between the heart and the dorsal aorta. It arises from the anterior end of the left ventricle. Three semilunar valves guard the opening into it, and a twofold mitral valve is situated between the left atrium and ventricle.

The subclavian and carotid arteries arise from the aortic arch. Their arrangement is not constant in different individuals but may take many forms. Brachiocephalic arteries very commonly occur.

The pulmonary artery arises from the right ventricle and divides into two, to supply the lungs. A ligament called the ductus arteriosus crosses between the aortic arch and the pulmonary artery.

The main branches of the aorta are:

1. The innominate arising from the top of the arch and dividing into the right subclavian and right common carotid arteries.

2. The left common carotid artery which arises from the arch just beyond the innominate, and forks at the angle of the jaw into internal and external carotid arteries.

3. The left subclavian, which arises from the left side of the arch.

The aorta bends to the left and runs posteriorly. Beyond the diaphragm it becomes the abdominal aorta. The main branches of this vessel are:

1. The coeliac, which arises just behind the diaphragm, and which divides into the hepatic to the liver and the lieno-gastric to the stomach, spleen, pancreas and duodenum.

2. The superior mesenteric artery, arising shortly behind the coeliac and going to the intestines.

3. The renal and genital arteries to the urino-genital organs.

4. The inferior mesenteric to the descending colon.

5. The two common iliac arteries which are the large terminal branches of the aorta, and which supply the limbs.

There is a complete double circulation. In the adult the two sides of the heart are completely separated. The left side contains only pure blood, the right side only impure blood. The atria contract simultaneously and the ventricles follow right afterwards. The impure blood is driven to the lungs, where it is oxygenated and returns to the left atrium by the pulmonary veins. The pure blood leaves the left

ventricle and passes into the aorta. From there it travels to all parts of the body. The three venae cavae collect the blood from the limbs, the head and the trunk and return it to the right atrium.

The lymphatic vessels are mostly gathered up into the thoracic duct. This opens into the left subclavian vein at its junction with the external jugular. The thoracic duct begins at the cisterna chyli and receives along its length the lymph vessels from the alimentary canal, the limbs of the left side, and the lower limb of the right side. There are numerous lymph glands in mammals but no lymph hearts. The tonsils are lymphoid structures and the spleen is midway between a lymph and blood-lymph gland.

The Development of the Circulatory System in the Human Embryo and Foetus.

The blood vessels originate in several scattered vascular areas, developed between the endoderm and mesoderm of the yolk sac, and later of the embryo. Certain groups of cells collect to form plexuses in which plasma collects, and a lumen or cavity is gradually formed. Some of these cells break loose from the wall of the lumen and become red blood corpuscles. Haemoglobin soon collects in them. All blood vessels are derived from these capillary plexuses.

At a very early stage two aortic arches form. These fuse to form a tubular heart, which consists of a sinus venosus, anterior to which is an atrium, a ventricle, the bulbis cordis, and most anterior the truncus arteriosus .

The vitelline veins in the body of the embryo and the umbilical veins from the placenta unite to form two ducts of Cuvier which lead into the sinus venosus. Two ventral aortae are given off from the truncus arteriosus. They divide to form six pairs of aortic arches which unite posteriorly to form the dorsal aorta. The circulation at this stage resembles that of the fish.

Later the heart forms an **S**. The atrium becomes partly divided by a septum and the sinus venosus then opens into the right portion of the atrium. The septum grows until it completely divides the atrium into two, then an opening called the foramen ovale opens through it. Another septum grows up through the ventricle to meet that in the atrium. The bulbus cordis becomes fused to the right ventricle and forms the infundibulum. The heart has now four chambers still incompletely divided. A septum divides the truncus arteriosus into the pulmonary artery, which fuses with the bulbus cordis, and the aortic arch which now meets the left ventricle.

Soon the pulmonary artery comes into communication with the sixth branchial arch, and the aorta meets the other five. The first and second arches degenerate. The third arches become the left and right common carotid arteries. The left fourth arch becomes the aortic arch, and the right fourth the subclavian of the right side. The fifth right arch degenerates, but that on the left side becomes the ductus arteriosus between the pulmonary artery and the aortic arch. This vessel exists until after birth. The left sixth arch gives rise to the pulmonary arteries, while the corresponding right arch atrophies.

Two groups of veins develop in the embryo, the visceral and the parietal. The visceral veins are the vitelline veins from the yolk sac and the umbilical veins from the placenta. These four open close together into the sinus venosus . The growth of the liver interrupts the growth of these veins, and causes them to break up into capillaries within its substance. The portion of the vitelline veins, which run on either side of the intestine, behind the liver, becomes the portal vein. The portion superior to the liver becomes the hepatic vein, which leads to the sinus venosus. The right umbilical vein disappears while the left enlarges and opens into the vitelline veins. With atrophy of the yolk sac the vitelline veins

atrophy. The ductus venosus forms from the right hepatic to the umbilical, so that blood from the placenta can go direct to the heart instead of through the liver. After birth the left umbilical and ductus venosus degenerate, and become ligaments.

The parietal group consists of two ducts of Cuvier leading into the sinus venosus. From the posterior part of the body two cardinal veins, and from the anterior part two primitive jugulars open into the ducts. The cardinals are formed by iliac and renal veins. The inferior vena cava is formed by the right cardinal up to the level of the inferior vena cava. Two subcardinals are formed, and later these give rise to the rest of the vena cava. The stage when cardinal veins exist is comparable to the venous circulation of a fish.

The jugulars enlarge to become the superior vena cavae. A branch joins the two jugulars, which becomes the left innominate vein.

The cardinal veins degenerate at the same time as the mesonephric kidneys, so that the renal portal system, which is quite comparable to that in the frog, disappears. The hepatic portal system, however, exists throughout the life of the organism.

BOOK REVIEW

THE PRACTITIONER'S LIBRARY OF MEDICINE AND SURGERY. 12 volumes and index. Price $10 per volume. (Only 8 volumes ready now). D. Appleton, Century Co., New York. 1932-1935.

This series of books attempts to present all the information that the general practitioner needs in the practice of any branch of medicine. A great number of very able writers have contributed articles in their special fields. The series is under the general editorial supervision of Prof. George Blumer of Yale. The success of his editing is shown by the remarkable uniformity of style and treatment seen throughout the many volumes. This series departs somewhat from the usual form of systems of medicine by attempting to be both more inclusive and more concise. In it are two unique volumes, the first which covers applied anatomy and physiology and the second which is devoted to the technique of clinical medicine. Both of these contain material which cannot be found collected together anywhere else.

In reviewing a series such as this it is impossible to adequately study all the volumes. However, after using the books for reference purposes for several weeks a few definite impressions have been formed. First, the books are unusually well printed, illustrated and bound and are arranged for easy reference. Second, the series appears to live up to its title and to present the most recent views on important subjects in which a general practitioner might be interested. The articles are concise, and devoid of all padding. In each case the available data has been carefully sifted and the writer presents his own considered opinion on the subject under discussion. The strict adherence to the scope indicated by the title has probably resulted in the exclusion of some material of interest to the student but not to the practitioner. For the recent graduate, who begins practice where no library is available, this set of books should prove very valuable. The publishers plan to issue a supplementary volume every two years to keep the series up to date.

O.M.S.

ABSTRACTS FROM CURRENT LITERATURE

It is hoped that the publication of abstracts of articles in the current literature, which are of special interest to students, will become a regular feature of the Journal. This first group of abstracts has been confined to publications by members of our own Faculty but in later issues material from other sources will be considered. Those who wish to find staff articles on subjects in which they are specially interested may do so by consulting the list of staff publications in the Medical Reading Room. Readers of the Journal, whether among the staff or the students, who encounter articles which they think would be of general interest to students, are urged to send in abstracts for this page.

Factors Causing Bronchiectasis and Their Clinical Application.

W.P. Warner, J.A.M.A. 1935, **105,**

Bronchiectasis is a condition in which the bronchial tubes are dilated beyond normal. In a thorough survey of 110 cases, 59% were found to have started with a known illness,—pneumonia and lung abscess being the most frequent offenders,—and this resulted in a chronic non-sepcific inflammation of the bronchial wall, destroying particularly the muscle and elastic tissue. Added to the infection is a physiological factor, namely dilatation of the bronchi in inspiration, due to 1. Pull of the expanding thorax; 2. Difference in pressure within and without the lumen. This dilatation is increased in the forced inspiration before coughing, and is rendered permanent by the inability of the damaged tube to resume its former shape.

Other contributing factors in the dilatation are,—1. *Atelectasis*—which increases the pressure difference, and transmits unmediated by the elastic parenchyma, the pull of the thorax. It also favors the accumulation of secretion, and hence furthers infection. 2. *Fibrosis of the Parenchyma*—with action similar to atelectasis. 3. *Central bronchial obstruction*—which results in a ball-valve action, so that both inspired air and secretion are retained.

Symptoms are commonly chronic cough and "chunky" sputum, influenced by posture and averaging about 4 ounces daily. It is foul-smelling in about 20% of cases. In the cases studied, haemoptysis occurred in 45%; 52% had a history of one attack of acute pneumonia, and 27% had had more than one attack following the onset of the bronchiectasis.

Signs are medium crackling rales, in the lower lobes, usually, and there may be physical signs of consolidation and fibrosis due to chronic bronchopneumonia. There may be signs of lobar atelectasis.

X-ray—plain plates are usually negative, but a triangular basal shadow is diagnostic of lobar atelectasis and was found in 16%. With iodized oil, the characteristic saccular dilatations are seen.

Prognosis depends on the parenchymal involvement and varied greatly in the cases studied, from 3 months to 42 years, the average being about 10 years.

Treatment. 1. Artificial pneumothorax during the acute infection to reduce the physiological dilating force.

2. Removing central bronchial obstruction where present and possible.
3. Postural drainage.
4. Surgical removal, only in the presence of considerable disability.

—J. F. D.

The Mechanism of Rotation in Occipito-posterior Positions.

John Mann, Can. Med. Asso. Journ. 1935, **33,** 607.

In this very interesting discussion of the mechanism of rotation in occipito-posterior positions there are two very novel features. The first is the suggestion that active movement on the part of the foetus is an important factor in producing rotation of the head as it descends in the pelvis. Considerable support for this hypothesis is brought forward and its practical implications in the management of labour are pointed out. The second novel feature is the description of the very ingenious forceps which the author has devised for rotation of the foetal head. In these forceps a universal joint makes rotation and extraction possible with one application of the forceps.

O. M. S.

The Importance of Rest and Liver Therapy in the Treatment of Subacute Combined Degeneration of the Cord.

R. F. Farquharson, Can. Med. Asso. Jour. 1935, **33**, 473.

This paper further emphasizes the excellent results which follow adequate treatment of subacute combined degeneration of the cord. The importance of giving ample liver and the advantages of the intramuscular injection of liver extract are pointed out. Patients showing definite signs of subacute combined degeneration are given the extract from 200 c. c. of liver intramuscularly, per week. This is double the usual dose for uncomplicated pernicious anaemia. Complete bed rest for at least three months is advised in all cases showing neurological signs.

The author concludes that, "The spinal cord lesions of pernicious anaemia can be prevented from developing and may be completely arrested by the administration of sufficient amounts of potent liver preparations.

Definite improvement occurs under treatment with prolonged rest and adequate liver therapy in those whose neurological symptoms are of relatively short duration."

—O. M. S.

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Coramine is a 25% solution of pyridine-b-carboxydiethylamide. By a direct influence on the centre, it acts as a most efficient respiratory stimulant; increasing both the depth and the rate of respiration.

Coramine stimulates the heart, leading to an increase in the output of blood; at the same time it tends to cause vascular relaxation, so that, with or without a rise in the blood-pressure, the rate and efficiency of the circulation are improved.

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The Clinical Diagnosis of Arteriosclerosis and Hypertension.

Duncan Graham, C.M.A.J. 1935, **32**, 29.

The Clinical Manifestations of Coronary Disease.

John A. Oille and Harold Rykert, C.M.A.J. 1935, **32**, 35.

The Treatment of Coronary Disease.

John Hepburn, C.M.A.J. 1935, **32**, 42.

This triad of articles on cardiovascular disease, appearing in the January, 1935 issue of the Canadian Medical Association Journal, should be of interest to all medical students, particularly at this University.

Professor Graham writing on 'Arteriosclerosis and Hypertension indicates the importantce of these factors in disease of the coronary arteries, which is the primary cause of heart disease in persons over forty. It is this group which shows the increased incidence of heart disease which now heads the list of the common causes of death. The article points out a common fallacy, namely, making a diagnosis of arteriosclerosis on the basis of sclerosed peripheral vessels. Such a diagnosis must be based on disturbances of function indicating impaired nutrition of various organs or systems of the body. The symptoms first become evident following increased activity of the organs. The pathological changes in all sizes of vessels; peripheral, renal, cardiac and cerebral are reviewed.

Attention is called to type of hypertension which is the cause rather than the result of sclerosis of small arteries and arterioles. Essential hypertension is at first probably due to increased tonicity of the arterioles. The pathological changes which follow are outlined. A brief differential diagnosis of cases showing increased systolic pressure is given, and the cardiac, renal and cerebral manifestations are described.

Dr. John Oille and Dr. Harold Rykert deal with 'the Clinical Manifestations of Coronary Disease" of which there are three groups. 1. Chronic degenerative myocarditis. 2. Coronary thrombosis and 3. Angina pectoris. This article is a statistical study with interesting comment on the variety of signs and symptoms presented. An analysis of a number of cases of coronary disease indicates the atypical pictures presented by cases showing complete absence of cardiac history, or in-

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farction without thrombosis, or thrombosis without pain. The value of the electrocardiograph in the diagnosis of obscure cases is indicated and the type of change found is briefly discussed. Dyspnoea, as the most important symptom of coronary disease, is discussed and classified into four types in order of severity. A brief differential diagnosis of chest pain, with a comprehensive description of anginal pain and of symptoms of cardiac infarction, is given.

Dr. John Hepburn discussess "the Treatment of Coronary Disease" under the heads: Degenerative Heart Disease without Cardiac Insufficiency, Degenerative Heart Disease with Cardiac Insufficency, Angina Pectoris, Cardiac Infarction. This paper is of particular interest because of the definite nature of the recommendations for treatment. The surgical treatments of Angina Pectoris, which have been developed in the past few years, are discussed and three case histories are presented which indicate the value of total thyroidectomy. A valuable discussion of Cardiovascular Tonics concludes the paper and, no doubt, will upset the ideas of many readers as to what tonics are really beneficial.

—B. H. McN.

The Role of Sympathectomy in the Treatment of Peripheral Vascular Disease.
R. I. Harris, Brit. J. Surg. 1935, **23**, 414

A review of the experience in the Toronto General Hospital for the past four years is presented. The importance of testing the dilatability of the peripheral blood vessels before operation is emphasized. The rise in skin temperature produced by spinal anaesthesia or nerve block is a reliable indication of the benefit which may be expected from operation.

In 24 cases of Buerger's disease the results were good in all but 5 cases. In these five cases the rise in skin temperature with spinal anaesthetic was less than 1°C. In a few selected cases of arteriosclerosis showing a rise of 3-10°C good results were also obtained. Cases showing a smaller rise in temperature gave poor results. Raynaud's disease of sufficient severity to demand operation is rare. In the four cases operated upon the results were not dramatic.

Royle's retroperitoneal approach, with complete removal of the lumbar sympathetic chain from diaphragm to pelvis is the operation of choice for the lower limb. For treatment of the upper limb the stellate ganglion is removed. Ramisection is not recommended because the results are less certain and less permanent.

—O. M. S.

Surgery in the Treatment of Pulmonary Tuberculosis.
Robert M. Janes, C.M.A.J. 1935, **33**, 389.

Interest in the treatment of pulmonary tuberculosis by collapse has been stimulated by the marked decrease in the mortality following operations for collapse and by the realization of the very short life expectancy of patients with cavities if collapse is not done. Pulmonary collapse may be brought about by (1) pneumothorax; (2) paralysis of the diaphragm; (3) scalenectomy; (4) pneumolysis, either intra-or extra-pleural; and (5) thoracoplasty. Of these pneumothorax and thoracoplasty are the most important and capable of the widest application. The indications for each type of collapse are briefly discussed.

—O. M. S.